

Tat-NR2B9c TFA

Cat. No.:	HY-P0117A	
CAS No.:	1834571-04-8	
Molecular Formula:	$C_{105}H_{188}N_{42}O_{30}C_2HF_3O_2$	
Molecular Weight:	2632.9	YGRKKRRQRRRLSSIESDV (TFA salt)
Sequence Shortening:	YGRKKRRQRRRLSSIESDV	
Target:	iGluR; NO Synthase	
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling; Immunology/Inflammation	
Storage:	Sealed storage, away from moisture	
	Powder -80°C 2 years	
	-20°C 1 year	
	* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)	

SOLVENT & SOLUBILITY

In Vitro	H ₂ O : ≥ 50 mg/mL (18.99 mM)					
	* "≥" means soluble, but saturation unknown.					
	Preparing Stock Solutions	Solvent Concentration	Mass	1 mg	5 mg	10 mg
		1 mM		0.3798 mL	1.8990 mL	3.7981 mL
		5 mM		0.0760 mL	0.3798 mL	0.7596 mL
		10 mM		0.0380 mL	0.1899 mL	0.3798 mL
Please refer to the solubility information to select the appropriate solvent.						
In Vivo	1. Add each solvent one by one: PBS Solubility: 100 mg/mL (37.98 mM); Clear solution; Need ultrasonic					

BIOLOGICAL ACTIVITY

Description	Tat-NR2B9c TFA (Tat-NR2Bct TFA) is a postsynaptic density-95 (PSD-95) inhibitor, with EC ₅₀ values of 6.7 nM and 670 nM for PSD-95d2 (PSD-95 PDZ domain 2) and PSD-95d1, respectively. Tat-NR2B9c TFA disrupts the PSD-95/NMDAR interaction, inhibiting NR2A and NR2B binding to PSD-95 with IC ₅₀ values of 0.5 μM and 8 μM, respectively. Tat-NR2B9c TFA also inhibits neuronal nitric oxide synthase (nNOS)/PSD-95 interaction, and possesses neuroprotective efficacy ^[1] .	
IC ₅₀ & Target	NMDA Receptor	nNOS
In Vitro	Tat-NR2B9c is a PSD-95 inhibitor, with an EC ₅₀ of 6.7 nM for PSD-95d2, representing a >100-fold higher affinity for this domain than for PSD-95d1 (EC ₅₀ , 0.67 μM). Tat-NR2B9c inhibits NMDAR2A, NMDAR2B, and NMDAR2C binding to PSD-95, with IC ₅₀ s of 0.5 μM, -8 μM, and 0.75 μM, respectively.	

	Tat-NR2B9c also blocks the interaction between PSD-95 and nNOS with an IC₅₀ of -0.2 μM^[1]. Tat-NR2B9c reduces association of PSD-95 with GluN2B by -50% in the YAC128 striatum, decreases NMDA-induced p38 activation in YAC128 striatal tissue, but shows no effect on the NMDA-induced JNK activation^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Tat-NR2B9c (10 nmol/g, i.v.) reduces infarction volume of male C57BL/6 mice, but has no effect at 3 nmol/g^[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Cereb Blood Flow Metab. 2019 Aug;39(8):1588-1601.
- Sci Rep. 2018 May 18;8(1):7848.
- Behav Brain Res. 7 January 2022, 113537.
- J Neuropathol Exp Neurol. 2020 Jul 1;79(7):800-808.
- PLoS One. 2020 Mar 3;15(3):e0229499.

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REFERENCES

- [1]. Cui H, et al. PDZ protein interactions underlying NMDA receptor-mediated excitotoxicity and neuroprotection by PSD-95 inhibitors. J Neurosci. 2007 Sep 12;27(37):9901-15.
- [2]. Fan J, et al. P38 MAPK is involved in enhanced NMDA receptor-dependent excitotoxicity in YAC transgenic mouse model of Huntington disease. Neurobiol Dis. 2012 Mar;45(3):999-1009.

Caution: Product has not been fully validated for medical applications. For research use only.

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